peritoneal cavity occurred, to produce either local or general peritonitis. As in other tissues, organisms may pass into surrounding spaces without gross evidence of an opening for their escape.

Complications.—(1) Paralytic ileus, 11 per cent; (2) pneumonia, 8 per cent; (3) subhepatic abscess, 3.5 per cent; (4) pelvic abscess, 3.5 per cent; (5) acute suppurative parotitis, 3 per cent; (6) fistula requiring closure, 2 per cent; (7) empyema of pleural cavity, 1 per cent.

In cases with perforative appendicitis the following complications were found. Eleven per cent developed paralytic ileus and most of these recovered when treated with a duodenal tube, intravenous saline, and heat to the abdomen. Pneumonia occurred in 8 per cent after operation and most of these recovered. Subhepatic abscesses developed in 3.5 per cent and pelvic abscesses in 3.5 per cent. The very unpleasant complication of acute suppurative parotitis occurred in 3 per cent. Fistula through the operative incision requiring closure developed in 2 per cent, and empyema of the pleural cavity in 1 per cent. Of all the patients in whom these complications developed only 15 per cent required second operations for drainage of abscesses, etc.

Death rate.—(1) From general peritonitis, 65 per cent; (2) paralytic ileus, 8 per cent; (3) pneumonia, 7 per cent; (4) subphrenic abscesses, 5 per cent; (5) pylephlebitis, 4 per cent.

In the first five years included in these records the mortality rate in cases of non-perforative appendicitis was 3 per cent. When, however, the cases were not treated until perforation had occurred there was an appalling death rate of 18 per cent. The causes of death in this group were general peritonitis in 65 per cent, so-called paralytic ileus in 8 per cent, pneumonia in 7 per cent, subphrenic abscesses in 5 per cent, pylephlebitis in 4 per cent and in the remaining group it was difficult to determine the precise cause of death. In the last five years covered by these records the mortality rate has dropped to 13 per cent, which is a distinct improvement. The cause for this change in death rate is rather difficult to assess, but possibly the increased interest and diffusion of information about the subject has educated the public so that they are aware of the dangers of the disease and demand earlier treatment. Undoubtedly the medical profession is learning more about the disease and appreciating the vast difference in results obtained by operating on cases before perforation has occurred as compared with those following perforation. It is with the object of further emphasizing these points that this analysis of symptoms and signs has been undertaken, and the importance of certain features of appendicitis has been pointed out. No attempt has been made to suggest treatment for appendicitis apart from early operation, as the object here has been to encourage early diagnosis and institute proper treatment in the hope that complications may be avoided.

THE RELATIVE VALUE OF THE DIFFERENT ESSENTIAL PHASES IN THE WHITE CELL AND DIFFERENTIAL COUNT IN THE DIAGNOSIS OF APPENDICITIS

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AS a prelude to the subject of this article, we observe that of the 400 cases of appendicitis (proved to be such by biopsy), dealt with herein, all gave some indication of the condition in at least one aspect of the blood count. Many cases resembling appendicitis clinically, were ruled out by entirely negative blood examinations. In the fæces of some of these patients, pin-worms were found which presumably were causing irritation in the region of the appendix; in other cases positive agglutinations were obtained with micro-organisms of the gastro-intestinal infections.

In the period over which these observations were made, 1932 to 1936, the average for each month of the counts, in proved appendicitis cases, were graphed* showing the average per

* Averages in white cell and differential counts of the percentage of polynuclears, lymphocytes, neutrophils with unsegmented nuclei (modified Schilling count), and total number of white cells, estimated monthly over a period of 4 years, 1932 to 1936, covering 400 authentic cases of appendicitis.

month of the total white cell count, the relative percentage of polynuclears, the percentage of lymphocytes and the percentage of non-filament cells (modified Schilling count). Cases of appendicitis in children under 10 years of age are left out.

It will be seen from the graph that the average total white cell count is as low as 10,000 per c.mm. in the month of October (•).

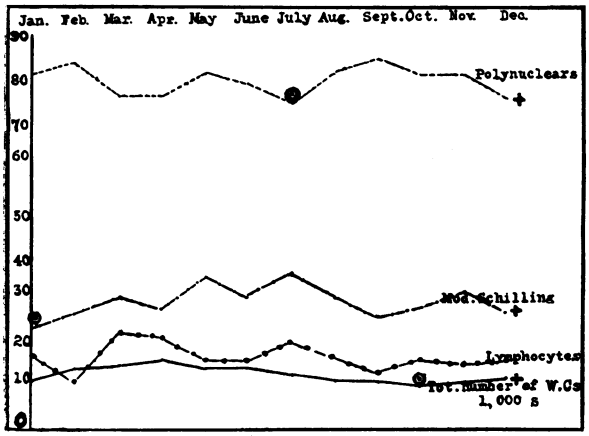

Chart 1

The polynuclear percentage is only 74 for the month of July. The non-filament cells show the lowest average in January, which is still high enough to indicate a definite moderate degree of inflammation. Taking the averages for December (+), we see total white cells 11,000; polynuclears average 76 per cent; both of which are of dubious import; but the non-filament average is 27 per cent — a fairly definite index.

At certain seasons of the year, particularly late winter and spring, many cases of appendicitis follow a gastro-intestinal, or influenza-like infection, which is accompanied by a leucopenia and a high relative percentage of lymphocytes. When two or more blood counts are done on such patients, an indication may be given of their condition in the increase of neutrophils, from normal or subnormal, rapidly to above normal, but usually in the first count the increase in non-segmented forms gives warning of an inflammatory condition.

The following counts from an actual case clearly illustrate these points:—

The patient had been sick for 10 days with a gastro-intestinal condition diagnosed as "gastric influenza".

August 4, 1938.—White cells, 7,150; differential white cell count: polynuclear neutrophils, 80 per cent, lymphocytes, 20 per cent. Modified Schilling count: 54 (severe).

August 5, 1938.—White cells, 10,450; differential white cell count: polynuclears, 83 per cent, lymphocytes, 17 per cent. Modified Schilling count: 62.

August 7, 1938.—White cells, 14,300; differential white cell count: polynuclears, 87 per cent, lymphocytes, 13 per cent. Modified Schilling count: 61.

Operation showed ruptured appendix.

The modified Schilling count on the 4th gave ample warning of what was to be expected. No very definite conclusions could be drawn from the total white cells or polynuclear percentage, until the second count, when an increase of 3,300 white cells and 3 per cent in the relative polynuclear count was recorded.

The following is a typical count on a case showing severe pain in the right lower quadrant; due to parasitic irritation.

Total white cells: 6,000. Differential white cell count: polynuclears, 53 per cent; lymphocytes, 30 per cent; large lymphocytes, 10 per cent; eosinophiles, 5 per cent; mast cells, 2 per cent.

In this case ova of oxyuris were demonstrated in the fæces.

In examining our results, we find a definite series in which the total white cell count is high, and in which the modified Schilling does not seem to be in conformity with this high white cell count.

The following actual case may be taken as a type for the series:

White cell count: 20,000. Differential white cell count: polynuclears, 84 per cent; lymphocytes, 10 per cent; large lymphocytes, 6 per cent. Modified Schilling, 21.

Operation showed appendix tense and filled with pus.

It seems from this that there is a threshold in relation to the white cell increase at which a mechanism comes into play to modify the influx of new neutrophils to the blood stream.

On the whole our experience shows that the modified Schilling, as we have applied it, has been the most useful phase in the white cell estimations.

We suggest that, where the enumeration of the white cells is impracticable, smears could be taken from the patient for differential white cell and modified Schilling counts.

Since preparing this article I have read with interest, the article by Watson and Sarjeant.[1] We had accepted the standards of normal of Prof. Schilling, but realized from the article referred to that a great discrepancy may result from using different techniques. Consequently

I took a group of 50 normal people, volunteer blood donors and others, and found the non-filamentous cells per 100 white cells averaged 8.6 per cent for the group. This confirmed our assumption that, if a patient shows a modified Schilling count of 15 or over we suspect some abnormal neutrophilic response.

REFERENCE

1. WATSON, C. H. AND SARJEANT, T. R.: Significance of low leucocyte count in acute pyogenic infections, *Canad. M. Ass. J.*, 1938, 39: 460.

TORSION OF THE TESTICLE

(WITH REPORT OF A CASE)

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TORSION of the testicle is commoner than is usually realized or than one would imagine from the number of cases reported in the literature. It is therefore not unworthy of a short discussion, as every practitioner will probably see two or three cases.

Etiology.—Torsion never occurs in a normally developed and situated testis. There must always be some degree of underdevelopment or abnormal development. Owen has demonstrated that a testis in its normal position in the scrotum, with a normal mesorchium, cannot undergo torsion.

Numerous developmental factors have been described as contributing to this condition; we shall briefly enumerate a few of them. A disproportionately capacious tunica vaginalis is a common finding.[1, 2] Campbell[3] found the gubernaculum testis to be unusually long and lax or absent in 8 of his 15 cases. Other factors described are elongation of the globus minor, considerable separation of the epididymis from the testis, and an abnormal attachment of the mesorchium to the lower pole of the testis, resulting in a more horizontal position of the testicle in the scrotum.

Assuming the presence of one or more of these abnormalities, any slight exertion may be sufficient to cause torsion. Coughing, sneezing, straining at stool, have been the initiating factors in various cases, but the torsion may occur during sleep. Uffreduzi states that the initiating mechanism is a spasm of the cremaster.

Torsion may occur in a fully descended testis or in an ectopic testis, as this latter is usually very mobile. In Uffreduzi's series of 80 cases, 60 per cent of the torsions occurred in undescended testes. Torsion usually occurs during adolescence, the average age being 17.7 years, but it may occur in early infancy or in middle age. No cases have yet been reported of torsion occurring in old age. In O'Conor's[4] series of 124 cases, 70 involved the right testis and 54 the left. A few bilateral cases have been reported.[4, 5]

While the vast majority of cases are acute and result in destruction or removal of the testis, a few cases are subacute and recurrent. The attacks are relieved by detorsion, either spontaneous or manual.[5, 6, 7]

Pathology.—The torsion may occur either outside the tunica vaginalis or inside it, the latter being by far the commoner in the descended testis. The degree of torsion may be anything from a part of one revolution to several complete turns. The direction of rotation is almost always the same; clockwise on the left side, and counter-clockwise on the right.[5] The skin and subcutaneous tissues are thickened and œdematous. The tunica vaginalis is infiltrated and contains a thick, almost black, fluid. The testicle and epididymis are swollen and plum-coloured, and it may be impossible to distinguish them by palpation. Gangrene rapidly occurs from strangulation, and hæmatogenous infection may result in suppuration. If untreated the mass is eventually completely absorbed. In the recurrent cases there may be no changes whatever, but there is usually a greater or lesser degree of atrophy.

Symptoms.—These will of course differ according to the degree of strangulation, and may be very slight in the recurrent cases. In the typical acute torsion they are usually very severe. The onset is sudden, with agonizing testicular pain, which may be accompanied by nausea and vomiting and even severe shock. There may be reflex abdominal cramps suggestive of intestinal obstruction. There is a